THE PLANT DISEASE REPORTER

Issued By

CROPS RESEARCH DIVISION

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

BLUE MOLD OF TOBACCO

Supplement 250 June 15, 1958

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THE PLANT DISEASE REPORTER

MYCOLOGY AND PLANT DISEASE REPORTING SECTION

Crops Research Division — Plant Industry Station, Beltsville, Maryland

BLUE MOLD OF TOBACCO

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INTRODUCTION

The blue mold, or downy mildew, disease of cultivated tobacco (Nicotiana tabacum L.), caused by Peronospora tabacina Adam, has had a long and colorful career. It might be pertinent to begin the discussion of this disease with a review of its history and of how it got its name.

In 1863 De Bary (33) reported a fungus in Europe on henbane (Hyoscyamus niger L.) which he named Peronospora hyoscyami d By. Then, in 1885 when Farlow (37) was traveling in California he observed a fungus growing on the leaves of a native plant, Nicotiana glauca Grah., around San Diego. Although this was a common roadside plant from San Diego to Los Angeles and Santa Barbara, Farlow could not find it at Santa Barbara. He did not have opportunity to observe farther south; therefore it is possible that it might have occurred even as far south as Mexico. After microscopical examination the disease was determined to be caused by Peronospora hyoscyami and was characterized by large grayish-black spots on both sides of the leaves, up to 2 inches or more in diameter, with an irregular but sharply limited circumference. No oospores had yet been found in the species. Farlow expressed a fear that the disease might be transferred to a related genus, such as Hyoscyamus niger, and be carried north of the limit where N. glauca might grow but where N. tabacum is cultivated. In May-August of this same year of 1885 Harkness (45) reported a fungus on the leaves of N. bigelovii at Reno, Nevada which was identified as P. sordida Berk. Later, in 1914, P. sordida was also identified as the cause of a serious tobacco seedbed disease outbreak in Victoria, Australia (at the time this Australian record was the only one of this species observed on N. tabacum) (104). According to Stevenson and Archer (95), who in 1940 identified as P. tabacina Adam a fungus on N. attenuata Torr. west of Reno Hot Springs, Nevada, both the disease on N. bigelovii in 1885 and the one on cultivated tobacco in Australia in 1914 were also caused by the same fungus, the one causing the mildew on cultivated tobacco in the United States.

In Argentina in 1891 Spegazzini (87) reported a new species of Peronospora on living leaves of Nicotiana longiflora Cav. in the Palermo woods near Buenos Aires, collected on December 17, 1888. He described the species, including both conidia and oospores, and named it P. nicotianae Speg. He again reported its occurrence on N. longiflora near La Plata in the springs of 1891-95 and on N. sylvestris in September and October 1897 (88). In 1901 he observed P. nicotianae on a third host, living leaves of N. alpina, between Las Cuevas and Puente del Inca, in Mendoza in the Andes in March (89). Hauman-Merck (46) in 1915 and Hauman and Parodi (47) in 1921 wrote that the fungus described by Spegazzini on different native Nicotiana species appeared to be somewhat rare and of no economic importance in the areas mentioned in Argentina.

In 1923 Gäumann (38), according to Adam (3), "apparently without having had access to all the material, considered that the species of Peronospora recorded on Nicotiana spp., including N. tabacum, should be referred to P. nicotianae Speg."

In Victoria, Australia, in 1900 McAlpine (62) reported finding Peronospora hyoscyami on N. suaveolens Lehm. and also noted that N. glauca, which had been imported from America, was susceptible to attack. Also in Victoria, but 25 years later, Adam (1) publishing on control of the "Peronospora disease" affecting cultivated tobacco, reported the disease on both N. tabacum and N. suaveolens. According to Angell and Hill (10) in 1932 N. suaveolens is the original host of tobacco downy mildew in Australia. If one assumes that this host is a native plant, then the fungus also may be assumed to be native. However there are two other possibilities: either that the fungus was introduced with some species of Nicotiana, probably from South America; or that both N. suaveolens and the fungus were introduced together and the fungus then spread to cultivated tobacco. The first record of downy mildew from Australia was reported in 1890 from Queensland, although tobacco had been grown in that country for

about 60 years previous to that date. Angell and Hill also found that the blue mold fungus attacked seedlings of the following Nicotiana species: angustifolia, atropurpureum, calicyflora, paniculata, campanulata, sylvestris, nudicaulis, repanda, caudigera, triangophylla, chinensis, glutinosa, acuminata, langsdorfii, laterina, and bigelovii. They concluded, therefore, that most, and perhaps all, species of Nicotiana were susceptible, but that Hyoscyamus niger was not susceptible. Also, they reported that the causal organism did not seem to fit descriptions of either P. hyoscyami or P. nicotianae, although it resembled the latter more than the former, and might possibly be a new species of Peronospora.

In 1933 Adam tried to resolve the nomenclature difficulty by the study of a Peronospora species on tobacco in Australia, which morphologically was very similar to both P. hyoscyami and P. nicotianae. He named this species P. tabacina Adam. Under artificial conditions this fungus was able to infect N. longiflora, the natural host of P. nicotianae; however it would not infect Hyoscyamus niger. He based his classifications chiefly on differences in the epispore and on oospore size.

In the United States in 1934 Wolf et al. (108) concluded that since it seemed quite unlikely that there were two species of Peronospora on Nicotiana, it seemed preferable at that time to regard the pathogen on cultivated tobacco as identical with P. nicotianae. In the following year, after the paper by Clayton and Stevenson (27), Wolf reversed this decision and accepted the name P. tabacina. Wolf et al. (108) increased the host range of the disease by observing symptoms on seedlings of tomato (Lycopersicum esculentum), pepper (Capsicum annuum), and eggplant (Solanum melongena), when these species were grown near tobacco seedbeds or in the same seedbeds with diseased tobacco seedlings.

At this period of investigation half of the problem had been solved. Adam had proved that the disease on tobacco could not be caused by P. hyoscyami, since that fungus would not attack Hyoscyamus niger, but there was still dissension as to whether the tobacco fungus should be called P. nicotianae or P. tabacina. The answer was given in 1935 by Clayton and Stevenson, who were the first actually to compare material from the Spegazzini herbarium with Adam's and Wolf's results. They concluded, among other things, that inasmuch as Spegazzini claimed that his conidia formed zoospores, and since the conidia of the tobacco fungus in this country have been germinated under many conditions and have never formed zoospores, it would not be advisable to call the tobacco fungus P. nicotianae. To quote, "P. tabacina, as described by Adam, on the other hand, fits our organism in all essential particulars; hence, the use of this name is recommended" (27). P. tabacina is the name now accepted by both Australian and American pathologists for tobacco downy mildew.

Three interesting cases of tobacco mildew occurring outside of the United States and Australia have been reported since 1935. In 1939 Wolf (106) reported receiving some diseased tobacco leaves sent to him in September 1938, collected in the State of Rio Grande do Sul, Brazil (an area not far distant from Buenos Aires, Argentina, where Spegazzini made his P. nicotianae collections). Sporangiophores were characteristic of Peronospora and within the leaf tissues Wolf identified glabrous oospores, which could not be distinguished in appearance from those of P. tabacina. He therefore identified the pathogen as P. tabacina and decided that both this species and P. nicotianae must exist in South America. The method of introduction into Brazil was unknown.

The second case involved a very serious attack occurring in the spring of 1939 on tobacco seedlings in the Lerma Valley (Province of Salta) and Perico (Province of Jujuy), Argentina (43). This was the first time that the disease had been found in Argentina since Spegazzini's time. For identification, diseased leaves were sent to the mycology section of the Spegazzini Institute of the National University of La Plata. The fungus appeared to be P. nicotianae, but there was some doubt as to identity as no oospores could be found on which to base the differentiation of P. nicotianae from P. tabacina. Identification rested on conidial size and on the size and shape of the sterigma of the conidiophores.

In the third instance, blue mold appeared for the first time in Cuba (48) during December 1957. It first occurred near Havana, over an extended acreage of "green-wrappers" type of tobacco. With much favorable weather during January 1958 -- overcast, rainy, a minimum of 59° F and a maximum of 71° F -- blue mold spread over a distance of approximately 100 miles. In the areas especially of Cuban cigar wrapper tobacco production the losses were high. In Havana Province where the disease first appeared there was considerable reduction in the normal production of green-wrappers. It remains to be seen whether blue mold will continue to be a serious problem on the island.

HISTORY OF THE DISEASE IN AUSTRALIA

Although it is patently impossible to trace the origin of a disease of such long-standing, it is now generally conceded that downy mildew is endemic to Australia. It is distributed throughout the tobacco-growing States of Queensland, New South Wales, Victoria, Western Australia, and South Australia. As far back as 1891 Cooke (30) reported the disease as being a "close ally of the dreaded mould of the potato disease," referring, of course, to late blight of potato, caused by another downy mildew fungus, Phytophthora infestans, which is also a Phycomycete and a close relative of the Peronosporas. In that same year the Agricultural Gazette of New South Wales (11) described the tobacco disease as a "blight.....closely allied to the notorious 'potato blight' ...., and is doubtless one of the most serious diseases of the tobacco plant." In 1911 Smith (84) said that blue mold had without doubt been the worst enemy of tobacco in Victoria and in 1914 (85) that blue mold "is possibly the greatest hindrance to quick development of the tobacco industry in Victoria." The first record of the disease in Western Australia was a disastrous outbreak on 14 August 1930 (73, 74) at Manjimup on plants growing on two adjoining properties. In South Australia in 1925 there was one instance of blue mold damage to fully grown tobacco leaves (78), but by 1935 such heavy losses were being sustained that some old growers temporarily ceased their tobacco production (39, 40, 41).

In addition to occurring on cultivated tobacco, downy mildew also is found commonly on Nicotiana suaveolens and other wild tobacco species, which serve as alternative hosts for the parasite. The prophecy of the early forecaster (11) who in 1891 declared about the disease that "It will grow worse and worse, year by year, unless means are taken to prevent it" was remarkably accurate. For downy mildew has grown worse, year by year, until today, about 65 years later, it has become the most widespread and serious disease of tobacco in Australia.

Tobacco downy mildew was considered such an important disease that in 1929 the Australian Government assigned cooperatively to Australian Tobacco Investigation and the Economic Botany Division of the Council for Scientific and Industrial Research (C. S. I. R.) the task of undertaking to learn the life history, the methods of over-wintering, and the processes of primary infection of the causal fungus (15). Work was begun in January 1929 and a staff for blue mold study has been maintained by the C. S. I. R. ever since. In 1934, at the unanimous request of producers associations, the government passed the Tobacco Growers Protection Act, aimed at eradication of all old tobacco plants in the field (40, 41). The provisions of this legislation will be discussed under the heading "Cultural Practices."

HISTORY OF THE DISEASE IN THE UNITED STATES

Downy mildew first made its appearance in the United States in four widely separated seedbeds of cigar wrapper tobacco in Gadsden County, Florida in the latter part of March 1921. By the beginning of April it had been reported from a number of seedbeds in the adjoining Georgia County of Decatur. Before the end of the growing season the disease had spread over all of the cigar wrapper area in the Florida-Georgia district. With the aid of pathologists from the United States Department of Agriculture (81, 82, 83) the disease was eradicated by burning diseased plants and by drenching seedbeds with formaldehyde solution. A weak Bordeaux mixture, 2-2-50, was recommended as a spray for healthy leaves. After this initial outbreak blue mold did not appear again until 10 years later, when it broke out with even greater vehemence than before, originating in the same Florida-Georgia area where it had first appeared in 1921. Before we discuss the spread of the disease from 1931 to the present it is advisable to spend a little time in consideration of how the disease happened to appear on cultivated tobacco in the United States, including the possibilities either that it was introduced into the country or that it is endemic.

A great deal of conjecture has arisen since 1921 in an attempt to explain the presence of this disease in North America. Regardless of the fact that all theories are still conjecture and probably must necessarily remain so, the evidence for both endemism and introduction from an extra-territorial source will be given for the record.

The case for endemism is rather strong and is based on various collections and observations by qualified investigators, principally Farlow's (37) collection on Nicotiana glauca in California, Harkness's (45) collection on N. bigelovii in Nevada, and Smith and McKenney's (81) report of the identification of Peronospora sp. in 1906 on cultivated tobacco seedlings at Halletsville, Texas. Since none of this material had oospores it is not possible to be certain of identification. However, in 1940 Stevenson and Archer (95) identified P. tabacina on N. attenuata collected in Nevada. In the spring of 1941 Godfrey (42) reported slight early damage

on pepper seedlings and also a downy mildew on wild N. repanda in the lower Rio Grande Valley. The causal organisms in both cases were subsequently identified as P. tabacina by J. A. Stevenson and E. E. Clayton. Then, in 1947 Wolf (107) identified P. tabacina which was occurring on several native Nicotiana species in Texas and Mexico, particularly on N. repanda. The sporangia and oospores were identical with those of the downy mildew on cultivated tobacco. Further, in 1949 Shaw (80) reported the collection in Washington State of a downy mildew on N. attenuata, by J. D. Menzies at Prosser, Benton County, in the summer of 1946. This specimen, identified as P. tabacina, contained oospores and is another reason for supposing that the earlier collections in the western United States were also P. tabacina. In view of his own and Shaw's collections and identifications, Wolf was of the opinion that the wild N. repanda, which grows profusely in the citrus and truck crop growing region of the lower Rio Grande Valley, was the source of inoculum for the outbreaks on tobacco in the Florida-Georgia areas in 1921 and again in 1931, and remained a potential reservoir of inoculum for the future. Thus it is suggested that P. tabacina is endemic to southern Texas and parts of northern Mexico, and that the gradual increase in tobacco acreage in the East has allowed the pathogen to progress from native wild western hosts to the more susceptible cultivated tobaccos of the East. This concept must take into account the dissemination by wind of the air-borne sporangia from the Rio Grande Valley to the tobacco areas of the East, a distance of about 1000 miles. The only fact that does not support the supposition of endemism to the Pacific Coast is the fact that one of the hosts, namely N. glauca, is not a native plant but was introduced from Argentina.

When downy mildew first appeared in 1921 several guesses were immediately advanced to explain its presence. Burger and Parham (18) thought that because almost all seedbeds affected exhibited the same symptoms at the same time, it was quite probable that the disease had existed, though undetected, in Florida for many years prior to the first general outbreak on tobacco. Government pathologists Smith and McKenney (81) considered three sources. They admitted the possibility that California and Texas might prove to be the original inoculum source, but did not try to follow up on the idea. They largely discounted introduction on infested seed in view of the fact that tobacco growers in the affected area either raised their own seed or imported only small amounts of seed from reliable Connecticut planters. The third avenue of approach was introduction from a foreign country.

Smith and McKenney believed that it was at least conceivable that the disease had been imported into the country on mats used in baling tobacco, such mats being brought into Florida from Sumatra. This seemed to be a natural starting place in tracking down the origin of the disease, particularly since the mats arrive second-hand after use on East Indian tobacco and also since it is known that certain insects have gained access into the United States in such a manner. Palm (69) hastened to refute such a claim. According to him the chances of infection from such a source are negligible when one considers the facts. The mats are manufactured from sedge grass in the plains of South Borneo, where apparently there is no tobacco cultivation. Then they are sent either directly to the United States or indirectly there by way of Holland. If the latter is the case, the mats first go to Java or Sumatra in the Dutch East Indies, where they are used for baling tobacco. After baling every package is disinfected for 48 hours with carbon dioxide and bales then go to Holland. The author knows of no genuine species of Peronospora having ever been found in the Dutch East Indies. During storage in Holland, while awaiting auction sales, tobacco from the Dutch East Indies might occasionally come into contact with tobacco from other countries, but the chance of contamination by resting spores which may adhere to the bales is very small. The only possibility remaining is contamination during shipping. Normally, every precaution is taken to store Sumatra and Java tobacco in rooms well separated from other cargo, chiefly to try to exclude contamination by the cigarette beetle Lasioderma; thus tobacco from other tropical countries such as British North Borneo, China, and British India also would be excluded.

DISTRIBUTION AND SPREAD

After its appearance in the Gadsden-Decatur area of Florida-Georgia in 1921, where losses were not over 5 percent (12), downy mildew was not again observed in this country until 1931, when it showed up first in the same region. This time it was much more severe and eventually spread to four additional States (90, 94), including the flue-cured areas of North Carolina and Virginia, and as far north as the air-cured regions of southern Maryland. It was also found for the first and only time in Louisiana, in St. James Parish. The disease may have been in South Carolina also that year; verbal reports make it probable, but there were no published

reports on its occurrence there (66). Despite prevalence of the disease in 1931, actual losses were slight owing to the confining of losses to seedbeds; and since growers had planted in excess of their requirements, commercial damage was far below the original estimate. The year 1932, on the other hand, was an epidemic one. Blue mold appeared early in the season in a seedbed at Tifton, Georgia. Soon it was reported from Florida and by the end of the season it extended its range to include South Carolina and Lancaster County, Pennsylvania, in addition to all of the States that had been invaded in former years (with the exception of Louisiana). There was a material reduction of the crops in Georgia and the Carolinas (91, 94), chiefly due to a shortage of transplants. Yield reduction in South Carolina that year was about 40 percent (17). Although the disease was destructive in 1933, it was not nearly so severe as in 1932 (92). Again it was common in Florida, Georgia, and South Carolina, and recurred in southern Maryland and in Lancaster County, Pennsylvania. But it spread farther into the burley area of central Virginia and was discovered for the first time in burley tobaccos in western North Carolina, southwestern Virginia and eastern and middle Tennessee. This was the first year for Tennessee. Apparently, also, this was a first record for the disease west of the mountains of North Carolina, eastern Tennessee, and western Virginia (94).

With the exception of the discovery of blue mold for the first time in 1936 in Kentucky, in a location not far from the Tennessee sites, the years 1934, 1935, and 1936 were characterized by a decrease in severity and in range of the disease. Losses in Florida in 1936 were confined principally to the cost of resetting about 5 percent of plants in the field (44).

In 1937 blue mold developed to epidemic proportions, as it had in 1932. In Florida about 25 percent of plants in beds were killed. The disease spread throughout the entire tobacco-growing region of Kentucky, was more prevalent in Tennessee, and once again appeared in western Virginia and western North Carolina, after an absence of 3 years. In Georgia, North Carolina and South Carolina practically every seedbed observed was affected and damage was high. In Georgia 60 to 80 percent of all plants were killed (26, 64). The mortality rate in South Carolina was greater than in Florida but much less than in Georgia. And in North Carolina there was more killing of plants than in South Carolina. This year marked the first occasion of destructive losses in the fields, rather than in just seedbeds or under shade. Blue mold appeared for the first time in Indiana and in the Connecticut Valley region of Connecticut and Massachusetts. In Connecticut it was not noticed until the late seedbed season when it caused considerable seedbed damage; and then in June it spread to the fields. However, hot weather in July caused the disease to disappear (4).

Figure 1 shows the distribution of blue mold in the eastern United States in 1921 and from 1931 through 1937.

Blue mold was comparatively mild in 1938. Although about 25 percent of the plants in Georgia were killed (26), in Florida the disease appeared later and did less damage than the previous year and there was no acreage reduction of flue-cured and cigar types tobacco (53). Blue mold was prevalent in the Old Belt of Virginia and North Carolina. It was found for the first time in Essex County in the Old Belt of Canada and was reported also for the first time in Miami County, Ohio.

In general, 1939 was a worse year than 1938 but not nearly so bad as 1937. In Florida infection came rather early and caused more damage than in the previous year (54) and in Georgia 50 percent of the plants were killed (26). In the years 1938-39-40 Massachusetts and Connecticut again felt the impact of blue mold, which reached the northern section of the tobacco-growing area, Franklin County, Massachusetts in 1938. In 1939 and 1940 Connecticut was affected in epidemic proportions. The Connecticut Experiment Station at Windsor estimated field losses in Connecticut on shade tobacco to be $100,000 in both years (5, 93).

Although blue mold has been found rather generally distributed each year since 1931 on tobacco in the Georgia-Florida area, in the Tennessee-Kentucky area it was not widespread until 1937 and from then until 1944 became less and less important until, in 1944, only two cases were observed or reported in this area. Valleau (97) believed that this would suggest that the fungus is not capable of maintaining itself north of the Georgia-Florida area for any indefinite period.

Annual losses for the years 1939 through 1946 were slight. There was considerable variation in distribution and severity, with no single really serious year until 1947, when winter temperatures in most localities favored development of the disease. However, in 1945 and 1946 the southern Ontario region of Canada had epidemic years. In 1945 the burley dark tobacco types of the old tobacco district of Essex and Kent Counties had at least 95 percent seedbed infection and there was some acreage reduction of burley either because plants were killed in the bed or because transplanting was postponed to allow for seedling recovery. In

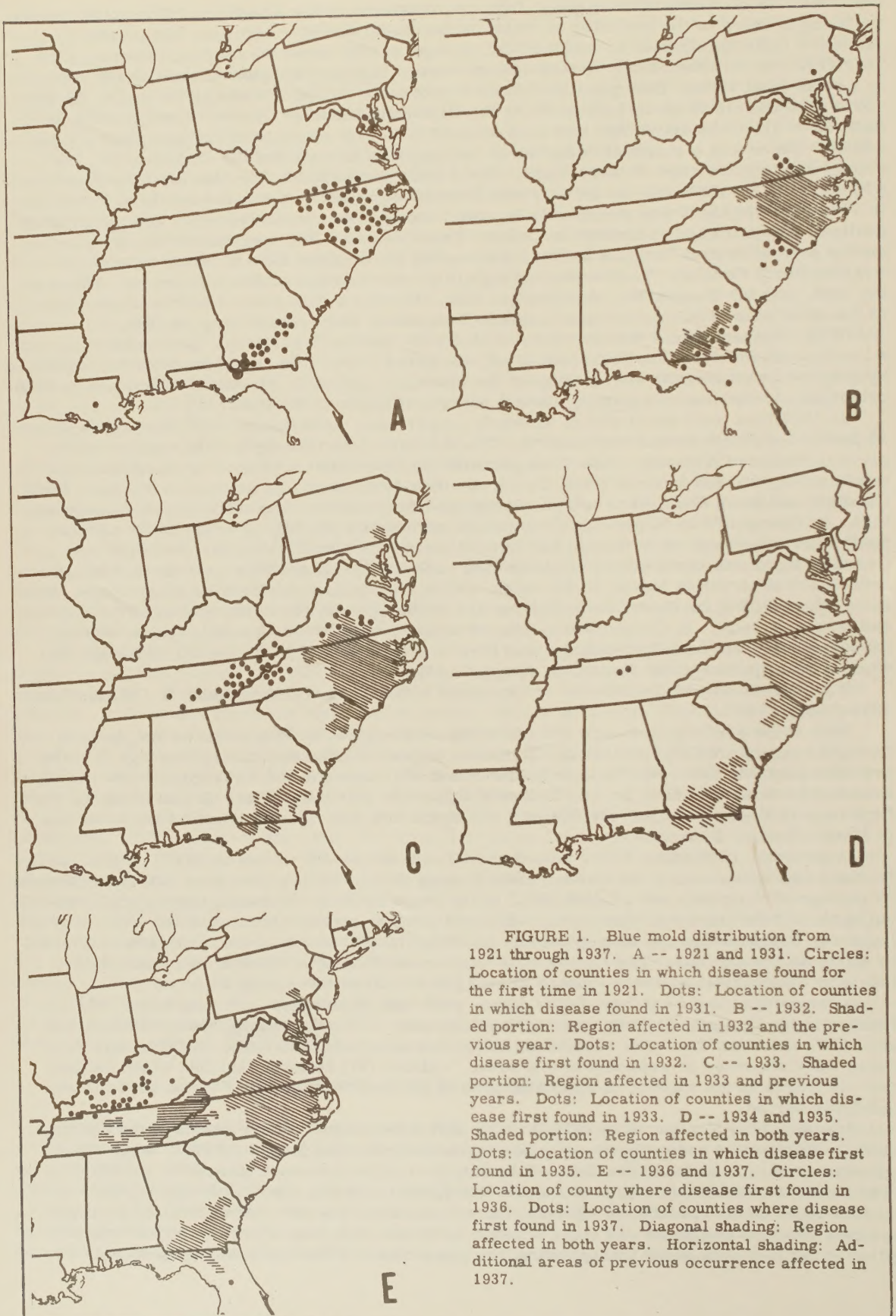

FIGURE 1. Blue mold distribution from 1921 through 1937. A -- 1921 and 1931. Circles: Location of counties in which disease found for the first time in 1921. Dots: Location of counties in which disease found in 1931. B -- 1932. Shaded portion: Region affected in 1932 and the previous year. Dots: Location of counties in which disease first found in 1932. C -- 1933. Shaded portion: Region affected in 1933 and previous years. Dots: Location of counties in which disease first found in 1933. D -- 1934 and 1935. Shaded portion: Region affected in both years. Dots: Location of counties in which disease first found in 1935. E -- 1936 and 1937. Circles: Location of county where disease first found in 1936. Dots: Location of counties where disease first found in 1937. Diagonal shading: Region affected in both years. Horizontal shading: Additional areas of previous occurrence affected in 1937.

1946 there was some spread to the field (96). In 1947 downy mildew came early to Florida, but owing to prompt application of fungicides little damage was done (56). Severity varied in seedbeds in Georgia, North Carolina, Virginia, Maryland, Tennessee, Kentucky, and Pennsylvania. The disease appeared in the field in South Carolina and was identified for the first time in July in Wisconsin, in a field of half-grown tobacco near the northern border of Dane County. A seedbed survey disclosed traces of infection on old plants which often were not too typical of blue mold; hence, it is possible that the disease had been present in Wisconsin before 1947 but had not been correctly identified (52).

From 1947 up to now blue mold has not been a major problem in this country either in the seedbed or in the field, owing principally to the use of protective fungicides. Although the disease was widespread in Florida in 1948, it usually occurred in isolated, scattered areas. In Georgia the overall plant loss was probably less than 3 percent. Blue mold damage was slight in 1949 except for a severe year in the Carolinas and Virginia. Although there was general distribution of the disease, on the whole 1950 was a mild year. The following year was also a light year. Average reduction in acreage yield ranged from 0 to 3 percent. Connecticut, on the other hand, had a loss of 10 percent of the value of the crop, or about $2,000,000 (68) in shade tobacco fields. The disease reached epidemic proportions in 1951 also in Ontario, Canada. The mildew, though general, was well controlled with fungicides in 1952. Although blue mold was widespread throughout the greater part of the tobacco-growing regions in 1953, plants were plentiful and there was practically no measurable damage. In 1954 North Carolina experienced the worst outbreak of blue mold in its history in the field and considerable damage was done in many fields in all stages of growth. The next 3 years, 1955-57, could be called light seasons, with a minimum of loss.

EPIDEMIOLOGY

January Temperatures

Damage from blue mold appears to be related both to earliness of infection and to temperature. In temperature observations over the years since 1931, when the disease first appeared to stay, a marked correlation had been noted between blue mold damage and late winter conditions, particularly January temperatures. Miller (66) in 1937 observed that in the peak years of infection, namely 1932, 1933, and 1937, the disease pattern in the southernmost tobacco-growing States of Florida, Georgia, South Carolina, North Carolina, and Virginia followed rather closely the deviations above normal of the corresponding January temperatures. In 1949 Miller and O'Brien (67) pointed out the correlation between the high mildew severity and high January temperatures in two additional peak years, 1947 and 1949. In 1956 the chart was extended to include the years 1950-1956. Thus, Figure 2 illustrates in graphic form just how closely disease incidence follows mean January temperatures in the over-all period of 1931-1956 in five southern U. S. locations. Although this relationship is not fully understood, one can assume that the warmer temperatures stimulate germination of the overwintering oospore at an early date which, in turn, allows for earlier infection and a build-up of inoculum and, consequently, of the disease potential. While a correlation of this nature cannot be considered a scientific foundation upon which to base forecasts, still it has displayed a very high percentage of accuracy and on this basis it is interesting to speculate on the outcome of a disease such as blue mold for, say, the current 1958 season. Because of the below-normal January temperatures that prevailed this year in the southern tobacco region, the prognosis is for late development of the disease and negligible damage. In addition, it must be borne in mind that even when subsequent temperatures during the growing season are favorable for development of the disease, actual damage usually is slight. Even in years when the high January temperatures approach the disease-initiating optimum of 62° F, unusually high temperatures during the growing period would modify the January temperature effect and might even be sufficient to nip an impending epidemic in the bud.

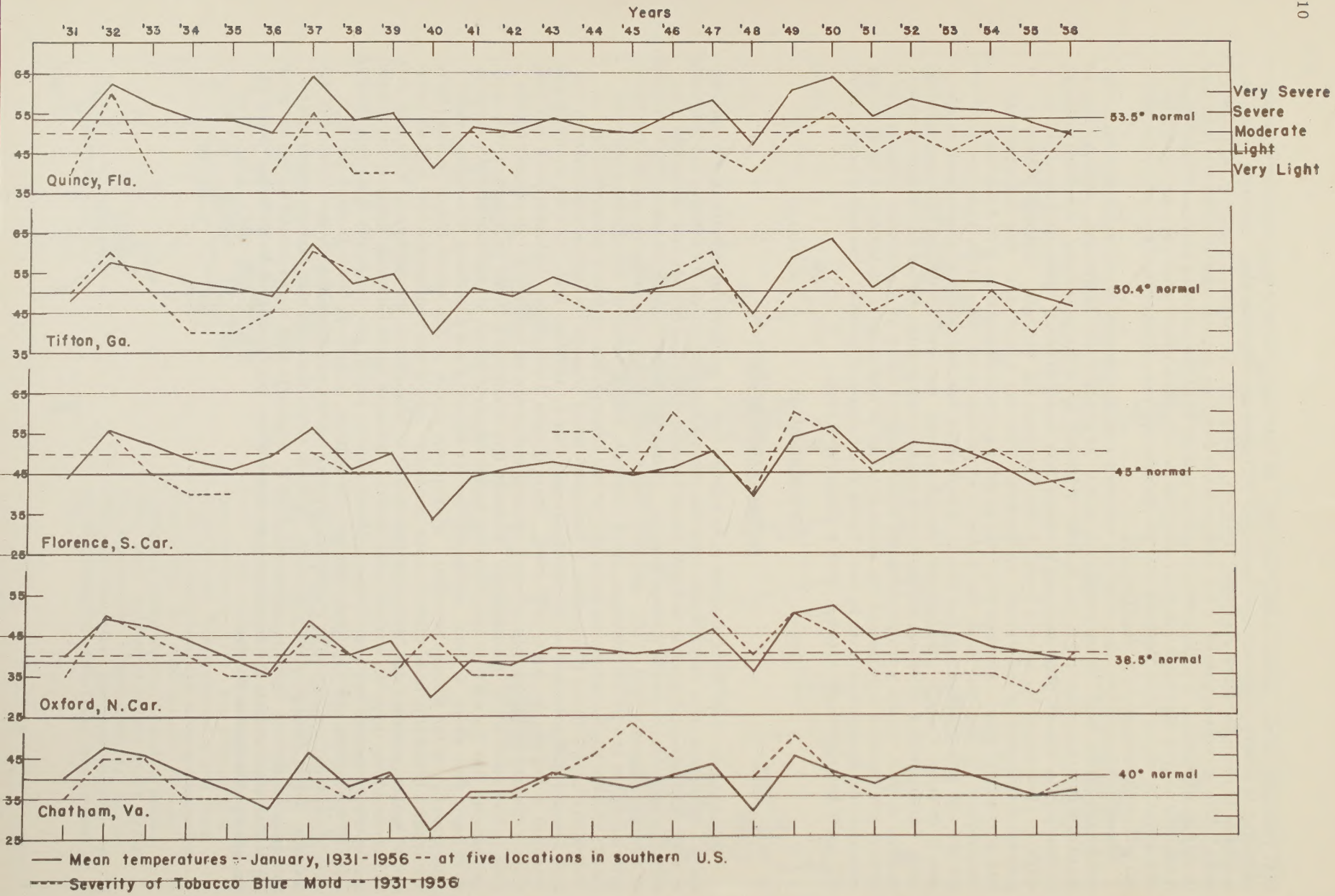

Figure 2. BLUE MOLD IN RELATION TO JANUARY TEMPERATURES

Source of Inoculum and Method of Dissemination: United States and Canada

In the United States and Canada both spore types of Peronospora tabacina can act as the source of inoculum. Primary infection usually is caused by the thick-walled overwintering oospores, while secondary infection results from germination of the thin-walled summer spores, called sporangia or conidia.

Under the heading "History of the Disease in the United States" a brief mention has already been made of the possibility that Texas served as the first source of inoculum for the blue mold outbreaks in 1921 and 1931 in the southeast. This theory came about in the following way. In early March 1947 Wolf (107) visited the lower Rio Grande Valley of Texas to observe blue mold on the wild tobacco species Nicotiana repanda. He found the fungus to be very common and at that time it was fruiting abundantly, which seemed to him confirmation that this region was the source of the disease in Georgia and Florida in 1921 and again in 1931, and that it would remain a potential source for the future. To follow up this idea, Valleau (98), in an effort to ascertain whether this same area in Texas could serve as an inoculum source for the Tennessee-Kentucky tobacco region as well, also visited this Texas area the following month, between April 17 and 30, 1947. At this time tobacco plants in Tennessee and Kentucky were far enough along to develop infection if given a source of inoculum. Valleau found, however, that the fungus had ceased sporulating on N. repanda in Texas, no doubt because of gradual increases in temperature between March and April. For this reason, and also because of the fact that blue mold had never been found in his area until 1936, Valleau quite logically concluded that it was unlikely that the Tennessee-Kentucky area would ever be subject to spore showers from the West and that the obvious explanation was that inoculum came from the southeast. He carried this a step further and said that if blue mold could be either eradicated from or greatly reduced in Georgia it would probably disappear from the Tennessee-Kentucky districts. This is a reasonable assumption when one remembers that the disease only became prevalent in the Tennessee-Kentucky area in 1937, following a general spore shower, and then it all but disappeared between 1938 and 1944. According to Valleau (99) there is evidence that in Kentucky the fungus cannot maintain itself year after year by oospores alone. After a spore shower the disease gradually disappears in succeeding years. Build-up of inoculum by oospores alone is slow and seldom results in an epiphytotic; consequently, the disease becomes less and less important as the fungus slowly dies out. Valleau (97) also explains that the unimportance of the disease in the Tennessee-Kentucky area from 1931-1937, when it was general in southeastern United States, is evidence that this area is not in a direct line of spread of spore showers from the South.

Some consideration has been given to the theory that the fungus might have migrated from the wild host N. repanda in the West to other wild hosts in the Gulf States. At the end of spring, when N. repanda dies in Texas, oospores may form in the leaves and pass the summer in dried leaf litter on the soil surface. These oospores are then able to infect the new rosette leaves of the host in late fall, thereby creating a continuous cycle of infection. There is no reason to believe that this happens in the southeast. The pathogen has never been found on a wild host east of the Mississippi. If such were the case, blue mold would have appeared before 1921, it would have appeared between 1921 and 1931, and the Perique area of Louisiana would have been affected more than just once. In the southeast the only known sources of primary infection in the spring are oospores that overwinter in dead leaves on the soil surface in beds used the previous year or the conidial stage of the fungus that overwinters on live tobacco plants, either suckers in the field or volunteer plants in old seedbeds. Plants living through the winter as a result of suckering have never been a major problem in this country. Volunteer plants sometimes do survive the winter in old seedbeds in the far South. During the winter of 1931-32 the fungus was found growing and sporulating on volunteer plants in Georgia; however, this is not very common. Most primary infection is the result of germination of one or a few oospores that have overwintered on the soil surface of old beds, the beds being used again the next season after either only disking or other shallow preparation. If, however, beds are plowed there should be no carry-over, because oospores must be on or near the surface in order to initiate infection. Although the fungus does overwinter on volunteer plants in southern States such as Florida, Georgia, and North Carolina, especially in mild winter years, farther north this does not seem to be true. In Connecticut, for example, the cold winters do not permit survival of host plants. Here primary infection results chiefly from oospores and very possibly also from spore showers blown from more southern States.

Primary Infection--Oospores: The relation of January temperatures to earliness of primary attack has already been discussed. Weather throughout the growing season is the limiting factor determining the progress and severity of the disease. Primary infection may occur regardless of rainfall. Dixon et al. (35) report that ideal conditions exist during or right after a period of warm weather, when temperatures have remained for several days at or above 50° F and when the lower leaves of the seedlings come in contact with the soil.

Primary infections are localized to one or at most to two or three spots in an occasional seedbed. Although thousands of oospores are formed, only an infinitesimal percentage is viable. This will be taken up in a later section. Oospores are believed to constitute the principal source of primary infection in the United States for several good reasons. First, the initial outbreak of the disease in a locality almost always occurs in old seedbeds (16, 34, 100, 101, 105). Second, there is always a lapse of about 2 weeks between the time infection is apparent in old beds and its first appearance in new beds (35, 100, 105, 109), and the fungus has been found sporulating on plants in old bed sites 7 to 19 days earlier than it developed in any new bed sites located nearby (34). Third, Dixon et al. (34) and Wolf et al. (109) in spore-trap experiments have shown that infections often develop before sporangia can be collected from the air in a given locality.

Primary infection in Canada is believed to have resulted from oospores in only a few years since blue mold first appeared in that country in 1938. A mild outbreak in the second season, 1939, apparently originated from oospore infection (29), as did also the limited infection in 1948 (96). Stover and Koch say that owing to the low incidence of overwintering oospores in Ontario it is not likely that a sufficiently high inoculum potential could arise from this source to create an epidemic.

Primary Infection--Conidia: Primary infection may also result from spore showers. As already mentioned, Valleau believed that the disease could not be maintained in the Tennessee-Kentucky area by oospores alone, but is dependent upon spore showers from the South. This probably is true also for the Carolinas and Virginia, with spores being blown in from the Florida-Georgia area. Maryland outbreaks, as well, are usually from such a source. Farther north, where oospore overwintering is not so common as in the South, spore showers account for much primary infection. Anderson (4) thinks that the first appearance of the disease in the Connecticut Valley resulted from wind-blown spores from Pennsylvania or States farther south. Subsequent outbreaks in Connecticut probably arose from overwintering oospores.

In 1952 Hyre (51) studied wind-dissemination of conidia as a means of primary infection. In an experiment to test long-distance spread of P. tabacina spores he grew a bed of tobacco on the University of Delaware Experiment Farm. Every possible sanitary measure was taken to preclude contamination by oospores of the seedlings, soil, seedbed covers, and seed. Blue mold developed. There was no tobacco cultivation within a radius of 20 miles, and the nearest tobacco beds (which were infected) were 20 air miles away in Pennsylvania and more than 20 miles away in Maryland. Hyre concluded that wind had borne the fungus spores over this distance of at least 20 miles to cause primary infection.

In Canada blue mold made its first appearance in 1938, the year that it spread to Ohio. In 1936 the disease came to Kentucky, where it became serious in 1937, spreading on into Indiana. Stover and Koch think it more than likely that the 1938 outbreak in Canada was the result of spore showers from Kentucky or Ohio, the tobacco-growing region of the latter State being only 125 miles southwest of Essex County, Ontario. A study of air currents during the critical period May-July revealed that the southerly winds in Ontario were of sufficient frequency to carry spores from the Ohio-Kentucky area to Ontario. Weather conditions were not favorable for development and spread of the disease in 1938, however. Between 1939 and 1944 the inoculum reservoir of the disease in Ohio-Kentucky declined and in Ontario the disease was unimportant, the isolated outbreaks being attributed to overwintering of the fungus in Ontario. Then, in 1945 the disease became epidemic throughout Ontario, and in 1946 it was nearly as bad. Correlatively, Ohio in 1945 experienced its worst blue mold year on record, and the same was true for Kentucky in 1946. Undoubtedly Canadian infection came from these two States. An abundant source of inoculum, plus favorable weather conditions, caused the disease to reach epiphytotic proportions. Another favorable year for development of blue mold was 1947; reduced damage this year was the result of widespread and effective use of the fungicide ferbam. To paraphrase Stover and Koch, widespread occurrence of the disease in Canada depends first upon the likelihood of primary infection by spore showers from the United States. To determine this likelihood it is necessary to know the distribution and severity of the disease in Kentucky and then, on the basis of wind direction and velocity, to estimate the chances of a spore shower. If the source of inoculum and wind conditions prove favorable, the second step

is to evaluate the importance of weather to spore viability, infection, and subsequent disease development.

Secondary Infection: Once primary infection has become established in an area, climatic conditions including rainfall, temperature, humidity, dew, and wind become all important in determining secondary infection and spread. Normally the number of sporangia borne on primarily infected plants is rather small. After secondary infection, however, the amount of inoculum increases to such a degree that within 10 to 14 days nearby beds will show infection. At the end of another week the amount of inoculum arising from these scattered beds is sufficient to cause a general outbreak, which in still another week may reach epiphytotic proportions by including all seedbeds within an area of several hundred square miles.

The causal organism of tobacco downy mildew requires coolness and moisture to complete its life cycle. The production and spread of sporangia may occur under the following types of conditions: low temperatures (40° -50° F) following a period of warm humid weather (23); continued cloudy rainy weather, especially when accompanied by winds (101); cool weather and plants wet with dew during much of the night and often late into the day (17); long periods of saturation when sky is overcast (35); very humid weather, followed by relatively warm days and cool nights (52). The disease is retarded by heavy rains and by warm sunny weather, but light rains favor development and apparently frost does not kill the spores (101).

Sporangia are formed early in the morning between daybreak and sunrise but usually only live a few hours if infection has not been initiated. They stick to the hands and clothing and are easily disseminated by wind. In fact, Clayton and Gaines (24) state that "At times the spores are carried long distances, as much as 50 to 75 miles or more; hence no seedbed in an affected area is likely to escape the disease, although some will be infected sooner than others."

In controlled temperature experiments in 1933 Clayton and Gaines (22) got good sporulation at temperatures ranging from 50° -65° F with an optimum near 60° -62°. There was no sporulation when the minimum remained about 70°, regardless of the maximum. In 1945 they found sporulation to be most abundant with a night temperature of 60° F preceded by a higher day temperature (25). In 1936 Dixon et al. (35) conducted extensive experiments on optimum sporulation conditions. They found that between the range of 42° and 63° F sporulation is abundant, with greatest production around 56°. Only a few spores were formed at temperatures below 42° or above 63°, and few or none above 68° or below 36°. Work reported from Virginia in 1938 agreed with these findings (101). However, in temperature-humidity studies in South Carolina in 1935 Armstrong and Sumner (17) obtained abundant sporulation at temperatures over the maximum named by Clayton and Gaines. On test plants maintained at 70° F and even a few degrees higher, sporulation was abundant. Today the optimum for sporulation and infection is considered to be about 62° F.

In most of the tobacco-growing regions of the United States temperature and humidity conditions favorable to the disease usually prevail throughout the seedbed period. However, in most years after seedlings are transplanted to the field, the temperatures soon become too high for the fungus to live, which probably accounts for the fact that blue mold in this country is primarily a seedbed disease and only becomes a hazard to plants in the field in years of unseasonable weather brought on by exceptionally late seasons. Such conditions are more apt to occur in northern tobacco regions than in southern ones, an example being the severe epiphytotic in Connecticut shade tobacco fields in 1951. Severe field outbreaks are the exception and normally northern regions conform to the general blue mold pattern. Anderson (4) says that even though the early seedbed weather in Connecticut may be too cold for development of the fungus, temperatures over most of the seedbed period favor the disease; then later, after transplanting, temperatures rise high enough to be prohibitive to further fungal development.

Source of Inoculum and Method of Dissemination: Australia

Downy mildew of tobacco is a far more serious disease in Australia than it is in the United States. While in our country the disease is confined principally to seedbeds and infection is limited almost entirely to leaves, in Australia most damage is done in the field and the disease is often systemic throughout the plant.

Climatic conditions favoring disease development appear to be similar to those in the United States. The production and spread of sporangia have been reported to occur under the following types of conditions: overcast or drizzly weather with reduced day temperatures for a period exceeding 24 hours (76); warm, humid daytime conditions and rather cold air temperatures at night, or fairly rapid alternations of warm, humid, and cold weather (75); severe outbreak often occurs during periods of dull, showery, and humid weather, or a succession of

fogs or heavy dews (61); appearance in the seedbed usually follows a period of excessive rainfall (13, 14); cold changes followed by muggy weather are conducive to the disease (84).

It is difficult to separate primary from secondary infection because the disease exists the year round, not just in the growing season (49). It has long been known that blue mold attacks the two wild species Nicotiana glauca and N. suaveolens. These species are widely distributed throughout Australia and consequently may be found wherever tobacco is cultivated.

Overwintering of the Fungus: The fungus may overwinter as oospores in dead plants, on living plants such as volunteers growing in sheltered places that remain cool or moist, or as mycelium in some part of the plant not killed by winter frosts.

Oospores are comparatively rare, but do sometimes occur (14, 31). In 1925 Adam found what seemed to be oospores in pieces of the bract present in seed heads of tobacco plants. During harvesting these pieces are mixed with seed and the disease is often perpetuated in this way.

The most common form of overwintering is as mycelium in wild hosts and in old tobacco plants which are not completely killed either by frost or by eradicative measures such as plowing after harvesting. In 1931 Adam (2) reported "Everything points to these old plants with blue mould being sources of infection in spring rather than diseased as result of infection in spring." In 1929 Darnell-Smith (32) indicated that, provided the weather is not too cold, tobacco plants may survive for 2 years, and that once a plant becomes infected it remains a potential source of infection. He cited the example of an infected plant that produced spore crops in November, the next March, and again the following November, even though the plant had been held in isolation. Angell and Hill (10) demonstrated the persistence of the disease in stems of plants remaining in the field over the winter; suckers arising from them in the spring showed blue mold symptoms, including sporulation of conidia, when the leaves were only a few inches high. Even if all of the aerial portion of the plant was killed by frosts, microscopic examination of sections of the shoots arising from underground dormant buds displayed the presence of the blue mold mycelium growing intercellularly in the young shoot tissues (50).

Although in the United States blue mold has never been demonstrated to be seed-borne, infested seed is a common source of inoculum in Australia. In experiments to test the seed-borne nature of the disease in 1929 Angell (7) grew seed from Australian sources and used as checks seed from North America. The Australian seed produced infected seedlings while the checks remained healthy until they were artificially infected by conidia from the diseased Australian seedlings. This work was followed up by Angell and Hill (10), who in 1932, upon examination of the ovules and seed produced in capsules taken from plants that had the fruiting bodies of the fungus on the flowers and capsules, found the typical coenocytic mycelium of the blue mold fungus.

In addition to infection by oospores and mycelium there is also the possibility that conidia may cause primary infection. Angell and Hill (8, 9) showed by experimental means that conidia may remain alive up to 2 months if kept cool and moist and about 5 weeks over fused calcium chloride in desiccators.

Spring Infection: Spring infection has been pretty well covered in the previous section on overwintering. The conditions affecting dissemination and spread of conidia by wind and rain are comparable with the United States. No area of any of the tobacco-growing States of Australia is free of the disease and, in contrast to the fluctuating pattern of the disease in the United States, blue mold in Australia is nearly always severe every year, although the degree of severity varies.

Effect of Temperature on Field Occurrence

Table 1 gives long-term mean temperatures and total precipitation for six locations in the United States and one location in Australia.

It will be noted that generally for each month of the growing season there is a difference of about 10 degrees in the mean maximum and mean minimum temperatures between the United States and Australia. Table 1 also indicates that the mean maximum temperatures in Australia do not rise much above 82° F, which apparently permits blue mold to continue to cause infection in the field throughout the season. In the United States, on the other hand, the mean maximum temperatures usually are high enough to inhibit the fungus from developing in the field. It would appear that when blue mold in the United States has become serious in the field temperatures have usually averaged 5 to 10 degrees below normal for the area involved. Perhaps this temperature-disease relationship can serve as a basis for predicting the occurrence and severity of blue mold in the field.

Table 1. AVERAGE TEMPERATURES AND RAINFALL FOR TOBACCO-GROWING REGIONS OF THE UNITED STATES AND AUSTRALIA.

Mo.	Hartford, Conn. 1921-1950			Chatham, Va. 8-year period prior to 1931			Chatham, Va. 1931-1952			Oxford, N. Car. 11-year period prior to 1931			Oxford, N. Car. 1931-1952		
	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.
Jan.	36.1	17.9	3.15	48.4	27.9	3.63	49.3	28.4	3.55	56.3	36.3	2.85	61.7	40.2	3.50
Feb.	37.9	18.2	2.57	54.0	31.9	2.86	53.2	32.6	2.99	58.0	37.2	3.30	64.9	43.0	3.64
Mar.	47.4	27.0	3.81	59.5	36.5	3.73	61.7	37.8	3.65	66.9	43.8	3.54	68.5	46.7	5.01
Apr.	59.9	36.0	3.56	69.2	44.1	3.42	69.8	45.4	3.79	75.2	51.6	3.67	78.7	54.7	4.31
May	72.3	47.0	3.66	78.2	52.6	3.77	77.0	53.0	3.89	83.2	59.3	3.78	84.4	60.9	3.40
June	80.9	56.8	3.62	83.7	61.1	3.75	85.1	62.6	4.52	90.4	67.6	4.88	88.8	68.0	4.11
July	85.6	62.0	3.56	88.8	66.4	4.75	88.1	66.2	5.63	91.1	70.6	6.45	90.0	70.1	6.50
Aug.	82.9	59.9	3.54	87.2	64.4	4.54	86.2	64.2	4.88	89.7	68.8	4.78	90.4	64.0	5.42
Sept.	75.5	52.1	3.44	81.8	59.4	4.37	82.5	60.7	3.69	86.1	64.4	4.28	88.4	67.2	3.44
Oct.	64.8	41.0	2.80	69.5	45.9	2.62	70.6	46.5	2.56	76.5	51.6	2.27	78.4	55.9	1.91
Nov.	51.4	31.2	3.48	58.3	36.9	3.05	59.4	37.4	3.16	65.7	41.4	2.28	68.1	46.0	1.96
Dec.	39.0	20.2	3.29	49.6	31.0	3.37	51.6	31.1	3.18	56.8	36.1	3.25	63.1	40.3	3.40

Mo.	Florence, S. Car. 1921-1950			Tifton, Ga. 9-year period prior to 1931			Tifton, Ga. 1931-1952			Quincy, Fla. 21- and 22-year period beginning 1931 (21)		Quincy, Fla. 1931-1952			Canberra, Australia 14-year period			Mo.
	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.	Mean Max.	Mean Min.	Total Ppt.	
Jan.	56.3	36.3	2.85	61.7	40.2	3.50	65.3	43.2	3.46	53.9	34.0	1.77	Aug.
Feb.	58.0	37.2	3.30	64.9	43.0	3.64	66.8	43.4	4.04	60.3	36.6	1.61	Sept.
Mar.	66.9	43.8	3.54	68.5	46.7	5.01	72.0	48.4	5.75	65.5	41.4	2.59	Oct.
Apr.	75.2	51.6	3.67	78.7	54.7	4.31	78.4	54.2	4.71	71.0	45.6	2.11	Nov.
May	83.2	59.3	3.78	84.4	60.9	3.40	86.4	61.6	4.30	78.6	50.8	2.18	Dec.
June	90.4	67.6	4.88	88.8	68.0	4.11	90.7	68.3	5.20	82.6	54.0	1.75	Jan.
July	91.1	70.6	6.45	90.0	70.1	6.50	90.4	70.0	7.06	82.6	53.7	2.07	Feb.
Aug.	89.7	68.8	4.78	90.4	64.0	5.42	90.2	69.9	6.69	75.4	49.8	2.10	Mar.
Sept.	86.1	64.4	4.28	88.4	67.2	3.44	87.6	66.4	4.49	66.8	42.8	1.87	Apr.
Oct.	76.5	51.6	2.27	78.4	55.9	1.91	82.1	56.7	2.23	57.7	37.3	2.86	May
Nov.	65.7	41.4	2.28	68.1	46.0	1.96	72.0	46.5	2.58	52.2	34.0	1.72	June
Dec.	56.8	36.1	3.25	63.1	40.3	3.40	65.9	42.8	3.98	51.5	31.7	1.46	July

*The 5 months enclosed in the box represent the growing season for tobacco.

ETIOLOGY

Oospores

According to Clayton and Stevenson (28) oospore size varies so greatly that it is of little taxonomic value other than to indicate the range for the genus, which is about 20μ to 60μ.

Because of the difficulty of finding oospores and, once having found them, of trying to make them germinate, very little is known about the factors involved in their formation and germination. Wolf and his colleagues (108, 109) have done most of the experimental work on this phase of blue mold; so the following information is based on their two papers. Oospores are formed within dead tissues and mature 4 to 7 days after death of the cells. A germ tube forms and enters the leaf either through the stoma or by penetrating directly through the epidermis, becoming intercellular mycelium with digitate haustoria that penetrate into the cells. In oospore-germination experiments, when pieces of decaying diseased leaves that had been collected in the spring and kept intermittently in cold storage and in the laboratory were macerated and put in drops of water on slides, a few oospores were seen to form germ tubes. In another experiment one portion of similar diseased material was air-dried and kept in the laboratory and the other portion was mixed with sand in a porous earthen vessel and buried in soil of an old bed. During the winter and spring of the following year samples were tested periodically in water drops to allow for germination at temperatures of 36° to 75° F. Less than a dozen oospores were seen to germinate in the tests which included several thousand oospores. When an oospore begins to germinate its contents become granular. The contents of the newly forming germ tube appear brown and granular at first, but as the tube elongates the contents become less densely colored. Length of the tube is about four times the diameter of the oospore. This is the only record of observed oospore germination in Peronospora tabacina.

In 1953, however, Person and Lucas (71) observed what at the time they believed to be oospore germination, not by germ tube, but by the development of sessile zoosporangia, which in turn produced motile zoospores. Later (72) they discovered that what they had seen had not been germinating oospores, but rather that the supposed zoosporangia attached to the oospores were actually a species of soil-inhabiting chytrid, Phlyctochytrium, that was parasitizing the spores. While this was rather a disappointing discovery to make, still it opens up a new field of investigation, namely, how important a role the chytrid might have in oospore germination.

Conidia

Blue mold conidia are ellipsoid, or egg-shaped, are very faintly violet tinted and, according to Clayton and Stevenson, are very variable in size. The range of means is 17μ to 28μ long by 13μ to 17μ broad. Like the conidiophores and oospores, the conidia have no distinctive morphological characters to aid in species determination.

The life cycle of the pathogen, including germination, penetration, incubation, infection, and sporulation, takes about a week. The sporangia or conidia form early in the morning and are completely mature by sunrise. They appear chiefly on the underside of the leaves, being borne on dichotomously branched conidiophores which emerge through the stomata. Waggoner and Taylor (102) experimented with a Hirst spore trap set 18 inches above the ground in a Connecticut tobacco bed. In one test they collected their first spores in quantity on a sunny, windy day at 4:30 a.m., with no spores after 8:30 a.m. On cloudy days spores first appeared at 6 a.m., and a maximum was reached between 9 and 10 a.m. Spore formation is favored by temperatures ranging from about 42°-63° F. As has been stated before, conidia are short-lived, usually remaining viable for only a few hours, and being killed by sunlight. Favorable conditions for infection are present if leaves remain wet for 2 to 3 hours after sunrise, for conidia must lodge on a film or drop of water to germinate.

If conditions remain favorable the sporangial cycle may be repeated in rapid succession. Clayton and Gaines (25) found that leaf infection by conidia was favored by 64° to 75° F. After penetration, the coenocytic mycelium grows intercellularly in the parenchyma of the leaf and also in the phloem region of the vascular bundles, forming haustoria which penetrate the cell walls (10). Within a week conidiophores, greatly branched, appear through the lower stomata and occasionally through the upper leaf surface just before dawn. Apparently invaded cells stay alive until sporangia are formed. Meanwhile the mycelium has thoroughly permeated all infected tissue, which results in eventual death of all cells in the vicinity, ordinarily after two or more spore crops have been produced. In the United States this necrosis is confined to the leaf tissue, but in Australia more often than not the mycelium sends out hyphae from the leaf

into the phloem, growing toward the center of the plant. When this axis is reached, hyphae grow both downward toward the root and upward toward the growing point to produce a systemic invasion of the whole plant. This is the mycelium that overwinters in any part of the tobacco plant left alive at the end of the growing season, thus creating a continuous source of inoculum.

SYMPTOMS

In the Seedbed

The very earliest symptoms in the seedbed are usually not very noticeable. If the weather is dry symptoms resemble nitrogen starvation. There is a slight yellowing of the tips of the leaves and small indefinite light spots may form on the under leaf surface. Later, leaves may become irregularly puckered or cupped and sometimes become twisted so that the lower surface is facing upward. If the weather is wet conditions are favorable for the disease to develop and there is rapid spread throughout the bed. Yellow spots appear on the leaves, which begin to darken and take on a water-soaked appearance. These spots may coalesce to form larger infection areas, on the under surface of which usually may be found the dense felt of "mold" which may be white, slightly bluish, or grey. This mold or mildew represents the fruiting bodies of the fungus and usually appears in the early morning but disappears later in the day; or it may not appear at all if the weather is dry. Severe leaf symptoms resemble scalding such as would result from the application of hot water or a toxic chemical like formaldehyde. Later the leaves dry up and flatten out into strings that lie on the surface of the soil. If beds have been closed, these other symptoms often are accompanied by the pungent odor of vegetable matter that has attained an advanced stage of decay, an odor not unlike that given off from potato plants rotting as a result of infection by late blight. On the other hand, if the blue mold attack is not very severe, very often tobacco seedlings will survive it, the symptoms will disappear, and after a sufficient period for recovery the plants can safely be set in the field.

In Australia seedbed symptoms are very similar, except that if wet weather favors the disease the fungus often goes on to attack leaf veins, either the subsidiary veins or both subsidiary veins and the midrib. Symptoms appear as dark-brown discoloration of the veins, visible from the under side of the leaf. Extreme attack of the midrib may even extend into the internal tissues of the stalk, causing sunken dark-brown blotches on the fleshy stalks.

In the Field

In this country field attacks are scattered and ordinarily are of relatively little economic importance except sometimes in peak blue mold years, when the disease becomes epiphytotic in the field as well as in the seedbed.

In the field entire leaves are not killed by the disease. The first sign of trouble is usually the appearance on the upper side of the lower leaves of yellowish indefinite spots about 1/2 to 1 inch in diameter. These spots often enlarge by coalescing, giving a more well-defined blotched area, often accompanied by leaf puckering, that soon begins to necrose and turn brown. When necrosis is complete these areas become papery and fall out, leaving ragged holes in the leaf that resemble either worm injury or Paris green burning. Leaf spotting is ordinarily confined to the lower leaves, being most severe on the sand lugs, many of which are discarded at harvesting. However, under severe disease conditions spotting may extend to leaves higher up on the plant. For example, Conners (29) tells us that in 1945, when the disease was severe in field tobacco in the old belt of Ontario, even the uppermost leaves of badly attacked plants were spotted. These spots differed from those on lower leaves in that they were bright yellow and did not become necrotic, indicating that infection was halted before the fungus was able to reach the fruiting stage. Person and Garriss (70) reported that during 1954, the worst blue mold year in the field that the State of North Carolina has ever experienced, leaf spotting extended up the plant frequently to the seventh and the eighth leaf and occasionally up to the fourteenth. On severely infected plants infection was heaviest on the bottom three to four sand lug leaves; many of these leaves exhibited 30 to 50 large necrotic spots and the entire area of some leaves was covered by as many as 70 lesions. In many fields this first priming of sand lugs, which represents 3 to 5 percent of the dollar value of the crop, was a total loss.

Despite an occasional scattered outbreak such as the one just described blue mold damage in the field in this country is not important in comparison with damage in the seedbed. In Australia a very different situation exists. In the early days of tobacco cultivation in Australia the disease was confined mainly to the beds, but now, except in Western Australia, where it

has remained chiefly a seedbed disease, practically all injury results from field infection. There are two types of field infection: localized leaf infection, called "spot mold"; and systemic infection, which may spread throughout the entire plant. Leaf infection symptoms resemble those in the United States. Leaves are infected progressively from the base of the plant upward. If the infection is very severe it spreads into the vascular tissue of the veins and ultimately may include either the stem or the roots or both. The ribs and stem show a dark discoloration and in the stem infection appears in the outer layers of the woody conducting tissue and in the inner layer of the "bark". Systemic infection sometimes kills the plant outright, but more often it causes a slow growth that results in stunting and uneven stands and leaf of inferior quality.

See Figure 3, A and B, for an illustration of symptoms on the tobacco plant. Figure 3B, provided by Dr. Howard E. Heggestad, has not been published previously.

CULTURAL PRACTICES

Plant Bed Management

Good cultural practices alone will not control blue mold once it has become established in a bed, but they can go a long way in helping to keep the disease out of the beds in the first place and in keeping it under control once it has arrived.

The following suggestions concerning good plant bed management represent the consensus of opinion of a majority of the people who have investigated the problems involved.

In the United States: Since the disease may be carried in the soil of the beds in the form of overwintering oospores, it is desirable, if possible, to select new bed sites every year. Beds should not be located on swampy land or near woods which create shade. An ideal location would be on eastern or southern slopes, to allow for protection from north and west winds and for maximum sunshine throughout the day. Soil must have good air drainage and ventilation, to permit fast run-off of water from soil and from leaves of plants. Valleau (99, 100) says that in Kentucky a system that works very well is to sow the beds after setting to a summer legume cover crop, which should be plowed under (not disked) in the fall. If neither of these two methods is feasible and the same beds must be used year after year for tobacco alone, there are certain elementary precautions that must be taken to prepare the bed for seeding. The beds must be sterilized, either with steam, by burning thoroughly, or by treatment of the soil with chemicals such as methyl bromide. If the seedbed is not to be used the next year, after transplanting it should be disked thoroughly or some other method should be used to destroy any hold-over plants that might seed and produce volunteers. This last recommendation is one of a list of items in a scheme for blue mold eradication presented at the Tobacco Disease Council meeting in Richmond, Virginia in January 1955. This scheme was adopted as a resolution and then was sent to the directors of the Florida and Georgia Agricultural Experiment Stations for any action which those States might wish to take.

Ever since blue mold became a threat to the tobacco-growing industry, it has been a common practice in southern tobacco States such as North Carolina to overplant seedbeds to ensure sufficient seedlings for a crop; where 50 square yards used to be planted for each acre to be set, it is now normal to plant 100 square yards per acre. Seeding of such "excess yardage" is probably a very good form of insurance for the grower, but such a precaution does not preclude the need for other methods of control. In spite of extra seedbed space, if conditions are right it is just as easy for downy mildew to wipe out 100 square yards as 50 square yards of young plants. When this happens growers must resort to buying seedlings from other growers, and in bad mildew years there are usually not enough to go around.

Seedbeds may be of any desired length but should be narrow to facilitate spraying, dusting, watering and weeding. Some growers recommend 4 yards or less while others maintain they should not be over 1 or 2 yards wide. Width depends upon a number of factors, the principal one being the type of equipment to be used in treating the beds. Once the bed has been prepared, the next consideration is seed. Although blue mold has not been found to be seed-borne in this country, it is a safety measure to use seed from healthy plants. Seed should be sown during the period recommended for a given area; however, beds should not be started excessively early, as such beds provide plants on which the disease can multiply and spread at an early date.

After plants emerge they must be protected from cold and wind by covers. However, it is important to know when the covers can be removed with safety. Removing covers during the day serves a two-fold purpose: it allows sunshine to harden the seedlings, and it reduces relative humidity in the beds. It is extremely important to keep relative humidity as low as

FIGURE 3. Symptoms and control of blue mold of tobacco. A -- Lower leaf surface view showing the "mold" growth characteristic of the disease. B -- Leaf spot phase of blue mold on shade grown cigar wrapper type tobacco. C -- Sprayed bed, which produced plants at the rate of about 40,000 per 100 square yards. D -- Unsprayed bed, which produced less than 1000 plants per 100 square yards. (Photographs taken from Tobacco Section, Crops Research Division, Beltsville, Maryland)

possible and leaves as dry as possible. In humidity experiments Armstrong and Sumner (17) obtained pretty good control of blue mold on larger plants by using forced-air ventilation--that is, by using a small electric fan to force air over a heating unit and then into the bed. Attempting to control humidity by such artificial methods is impractical because the impossibility of preventing dew formation on small plants makes them particularly vulnerable to infection. Maximum sunlight would seem to be the ideal solution. The importance of relative humidity in promoting infection is illustrated by infection of Perique tobacco in Louisiana. In 1931, when tobacco in St. James Parish was hard hit by blue mold, growers had covered their seedbeds with Palmetto leaves for protection from the cold. As a result humidity was kept high and the disease spread more rapidly than it might have under ordinary growing conditions (36).

Heat treatment of beds has been tried as a control measure, but has proved too expensive, requires too much labor, and does not work very well on small plants. Armstrong and Sumner found that minimum night temperatures of 88° F and above (that is, 88° to 93°) controlled the disease partly, but not completely. Clayton and Gaines (25) managed to stop sporulation with minimum temperatures of 70° and above; however, plants grown with reduced light, due to heavy shading in the greenhouse, sporulated freely at temperatures as high as 80°. Thus it would appear that there are too many factors modifying the effect of high temperatures to make temperature control practicable.

When plants are up in the beds it is good practice to water in the forenoon so the plants can dry off before night. Also, watering should be kept to a minimum because leaves should be kept as dry as possible and relative humidity as low as possible. If blue mold does appear in a bed the application of small amounts of nitrate of soda usually will promote recovery of the plant by stimulating rapid new growth of secondary roots near the base of the stem. On the other hand, too much of this chemical will make the plants too watery and succulent and consequently more susceptible. Improper application may even cause more damage than the disease itself (65).

Plants should never be set out in the field until they show definite signs of recovery, such as the formation of new roots, but there should be no delay in setting out as soon as the tobacco is large enough, has recovered from the disease, or both. After transplanting, any old plants in the beds should be completely destroyed.

In Australia: In Australia the same cultural practices are recommended with a few modifications and additions.

The problem of finding disease-free seedbeds is acute as the disease is not only widespread during the regular growing season but also exists many times over the whole year. If possible tobacco should not follow tobacco in a field, but should be rotated with other suitable crops. Beds should be isolated and should be sown in relays at intervals of every 2 to 4 weeks from August to November, so that some of the beds will miss the critical time for the moist season (13, 84). Soil should be fairly sandy and well drained and raised above the general ground level to prevent excess water from coming in contact with plants (60). Although it is difficult to keep beds sterile they should be treated before seeding by one of the following methods: steam at 100 pounds' pressure for 1/2 hour; burning for at least 4 hours with a wood fire; application of water with formalin at the rate of 1 gallon of formalin to 50 gallons of water (73).

Blue mold in Australia is often seed-borne; thus it is of primary importance either to use seed from guaranteed healthy plants or to treat the seed for about 5 minutes in absolute alcohol before sowing to kill any spores that may be adhering to them. In spite of the employment of the most rigid sanitary measures, it is often impossible to get a sufficient supply of disease-free plants for setting out. In fact, just before the discovery of the benzol gas treatment for seedbeds the Victorian Department of Agriculture arranged with the Department of Agriculture of New Zealand to have seedlings grown at Auckland and then shipped to Victoria when ready for setting out. This system proved not only to be very expensive but it was also a failure, as the plants usually became contaminated anyway.

Whenever infected plants are found in a seedbed they should be pulled up, roots and all, and burned, and after transplanting any plants remaining in a bed should be thoroughly destroyed. In Australia stress is also placed on the spread of the disease within a bed and between beds by people who carry spores on their clothing. For this reason visitors should not be allowed in any beds and workers should be very careful of contamination from this source. Insects, too, may be an important factor in disease dissemination. It has been demonstrated that the tobacco leaf miner, Phthorimaea operculella Zell. [=Gnorimoschema operculella Zell.], may transmit the spores, and it is possible that other insects also serve as vectors (61).

Other cultural practices such as allowing young plants plenty of air and sunlight, trying to keep beds warm enough so that they do not fall below 45° F, reducing watering to a minimum, delay of transplanting until seedlings show signs of recovery, and the use of narrow beds about a yard in width are the same as those reported in the United States.

Field Sanitation

When blue mold appears in the field there is not much that can be done in the way of sanitation except for removal and destruction of diseased plants and the plowing under or cutting down of all plants remaining after harvest so that suckers from the field crop cannot go into winter. This precaution was considered so imperative in Australia that in 1934 a new regulation was added to the Tobacco Growers Protection Act requiring the occupier of any land on which self-sown tobacco plants are or shall be growing to destroy completely all tobacco plants by fire or burial every year before the 31st day of July (40). Plowing is not considered entirely satisfactory since in some cases plants are covered by soil which protects them from frost and enables them to survive the winter. It is also necessary in Australia to destroy any plants of the native tobacco, Nicotiana suaveolens, or other wild tobacco such as N. glauca, which may be growing in a tobacco region. And, as with cultivated tobacco, the roots must be completely destroyed to be sure that no part of the plant remains alive which might harbor the mycelium of the fungus.

FUNGICIDES

Fumigation with Gases

In the latter part of 1934 and in early 1935 Dr. H. R. Angell of the Council for Scientific and Industrial Research of Australia was largely responsible for the perfection of a technique for blue mold control, which consisted of treating tobacco plant beds with vapors from benzol, an aromatic hydrocarbon distillate of coal tar. In the fall of 1935 extensive experiments were begun in the United States to test the efficacy of benzol. The benzol treatment consists of placing liquid benzol, at the end of the day, inside the beds in suitable glass or metal containers such as shallow pans or troughs (either with or without wicks) that are raised above the plants. Then the bed is covered with a heavy muslin material and the benzol allowed to evaporate during the night. The vapor, which is about twice as heavy as air, drifts down onto the plants, penetrates the leaves and kills the fungus, thereby acting as a curative rather than as a preventive measure.

In 1937 McLean et al. (63) in North Carolina confirmed the efficiency of the benzol method of treatment. They found that if lubricating oil were added to the benzol the partial vapor pressure was lower, rate of evaporation was less (owing to the higher viscosity of the mixture), and the concentration of vapor resulting was just right for inhibition of infection by the downy mildew organism but not high enough to injure the tobacco seedlings. Thus, the oil added the margin of safety necessary to make the benzol treatment "foolproof". Benzol was tried to a limited extent during the 1937 season, but was more widely employed during the next few years.

In 1938 Lunn and Mattison (59) reported from South Carolina that benzol completely checked downy mildew after it appeared in the beds. They did not recommend its use too highly, however, since it had a number of disadvantages. The technique is expensive and hard to handle. Benzol vapor is very flammable, it may be poisonous to humans, and if the benzol is splashed on plants it will either kill portions of the leaves or the entire plant. A great deal of labor is required to fill the evaporating pans every night and to remove the heavy muslin covers every morning and put them on again at night for the first three or four consecutive nights and then about twice a week during the period that blue mold remains active. Although the vapor produces a good bit of wilting in the middle of the day, the tobacco does not sustain any permanent harmful effects. Another advantage is that benzol vapor kills flea beetles and other insects which spread the disease within and between beds. Florida, Virginia, Connecticut, and Maryland reported the same general results. In Maryland benzol was considered impractical because the highly explosive nature of the material made it too dangerous to work with (103).

In 1939 a better method of fumigation was developed, when it was discovered that fumes given off from the evaporation of the solid white crystals of paradichlorobenzene (PDB), called "Paracide" or "Parabacco" commercially, are just as effective as benzol in controlling blue mold. The crystals are spread out on board shelves or in wire baskets built around the sides

of the bed or they are scattered on the surface of a thin cotton tobacco cloth stretched 8 to 14 inches above the ground, just over the top of the plants. This is done at sundown and then the entire bed is covered by a heavy, wet, gastight, muslin cover and the crystals allowed to vaporize during the night. In cooler weather more crystals are required as evaporation is slowed down by lower temperatures. The use of PDB was an improvement over benzol because the material was easier to handle, there was no danger of damage from spillage, application was faster since the necessity for liquid-evaporating containers was eliminated, and the crystals themselves were relatively cheap. The PDB crystal method was particularly welcomed in Connecticut, where the usual copper oxide sprays had not given satisfactory control, and so this treatment was commonly used there for a few years. The subsequent development of Fermate and other excellent protective fungicides has resulted in the gradual replacement by dusts and sprays of gases which, in comparison, are too cumbersome to use and constitute an unnecessarily expensive control precaution. However, PDB fumigation is still recommended for eradicating the fungus when it is already in the beds, if the grower has the right set-up and equipment for gassing airtight beds.

In Australia the situation is quite different. There, sprays of any type have not proved adequate for blue mold control and the benzol vapor treatment alone is advised for seedbed treatment (77).

Sprays and Dusts

When comparing sprays and dusts with gases it must be remembered that while gases act as eradicatives that can kill the fungus after it has infected the host, sprays and dusts, on the other hand, act as preventives and must be applied to the host plant before the disease appears.

When blue mold was first discovered in the United States Bordeaux mixture was recommended as the spray to use, partly because it was being used to some extent in Australia and partly because Bordeaux was the panacea usually suggested for the treatment of any new plant disease, particularly when nothing better was known. In the early 1930's, then, Bordeaux mixture was used, on and off, along with other tested fungicides, in strengths of 2-2-50, 3-3-50, 3-4 1/2-50, and 4-6-50 and it gave partial to good control depending upon many factors, the main one being whether it was correctly applied. It was fairly fungicidal in action, but too many sprayings often resulted in severe damage to the tobacco plant and in order to get "adequate control" it was necessary to obtain complete coverage of both leaf surfaces. "Adequate control" at that time meant that the development of the disease was delayed 1 or 2 weeks, because of delayed sporulation of the pathogen, and that usually when blue mold did appear the Bordeaux mixture minimized the severity of damage. However, in the case of a really severe infection, this fungicide was wholly unsatisfactory. In Florida it frequently caused an abnormal amount of stunting and retarded growth and it was found that Bordeaux mixture, as well as other copper-containing salts, interferes with the formation of secondary roots, probably as a result of the accumulation of the chemical around the base of the stem at the soil surface. In Australia Bordeaux formulas commonly used were 2-2-40, 2-2-50, and 4-4-40, as well as 3-3-50 Bordeaux mixture plus calcium caseinate, the latter to be used as a field spray.

Other fungicides used during this early period which gave a fair degree of control were red copper oxide-oil emulsion, colloidal copper, copper-soap, calcium monosulfide (Cal-Mo-Sul), lime-sulfur, and cuprous oxide and benzoic acid used with cottonseed oil. Most of these materials shortened the disease period, allowing for earlier transplanting, but none had any curative value. In 1938 in Queensland, Australia colloidal copper was fairly effective in the warmer parts of the State, but in the cooler parts the volatile gas method worked better; therefore it was recommended that a combination of the two systems by employed -- gases during cool, rainy spells, and sprays during warm sunny days. In this same year no sprays of any description did any good in Connecticut, while in Georgia red copper oxide gave rather fair control.

The development of the benzol and PDB fumigation techniques in the middle and late 1930's did much to bridge the gap between the hopelessly inadequate control job done by the aforementioned sprays and dusts and the excellent results afforded by certain organic materials, the first of which was tested in 1942. By 1938 the chief recommended controls for blue mold in the United States and Australia were spraying or dusting with red copper oxide or colloidal copper, and fumigation by the then recently perfected benzol method. In Australia sprays were far superior to dusts. Neither sprays nor dusts eliminated the disease, but they did reduce it to a point where it did little or no damage. In general, they seemed to give better control in the southern than in the northern tobacco regions of the United States, and even up to the early

1940's in Connecticut and Massachusetts the greater percentage of growers were still using the safe and sure gases.

The year 1942 ushered in a new era in fungicides for blue mold control. In May of that year Anderson (6) published results of extended greenhouse experiments on organic fungicides. He found that ferric dimethyl dithiocarbamate gave 95 to 100 percent control. And Kincaid (55) in Florida reported similar success with this new material in trials at Quincy, which resulted in excellent control, a little better than that with cuprous oxide-cottonseed oil emulsion. Another material, tetrachloro-para-benzoquinone, also gave good control, about equal to the cuprous oxide spray. (See Figure 3, C and D, for an illustration of the protection obtained with good organic sprays.)

Within a few years many other organic fungicides, a few of which proved to be very effective in blue mold control, were developed. They are as follows:

Common Name	Trade Names	Chemical Name
1. ferbam	Fermate Karbam Black Nu Leaf Ferradow Niagara Carbamate	ferric dimethyl dithiocarbamate
2. zineb	Dithane Z-78 Parzate zineb	zinc ethylene bisdithiocarbamate
3. nabam	Parzate	disodium ethylene bisdithiocarbamate
4. ziram	Zerlate	zinc dimethyl dithiocarbamate
5.	Bismate	bismuth subsalicylate
6. maneb	Manzate Dithane M-22	manganous ethylene bisdithiocarbamate

Table 2 shows the organic fungicides used in the various tobacco-growing States of the United States and in Ontario, Canada from 1945 through 1951.

From 1951 to the present the organics have continued to give satisfactory control, and particularly since 1948, when blue mold control was implemented by the establishment of a USDA Warning Service which notifies farmers in advance when to expect the disease in their vicinity. The two most widely used fungicides in the United States today for blue mold control are ferbam and zineb. These are the two chemicals most highly recommended by the United States Department of Agriculture.

Since about 1954 antibiotics have been investigated as a means of blue mold control. The degree of control seems to have been variable. Generally, streptomycin sulfate formulations at the rate of 200 parts per million have given good results. This material has been extensively and successfully used in the burley-growing sections of Tennessee, Virginia, and western North Carolina for the simultaneous control of both blue mold and wildfire.

In Australia the fungicide picture is very different. As already mentioned, the benzol treatment is used almost exclusively in the seedbeds. And in the fields it is very difficult to control blue mold with sprays because there must be timely and effective coverage of both top and bottom leaves of new foliage, which is naturally expanding rapidly at this stage of growth (76).

RESISTANT VARIETIES

During an attempt in 1934-1935 to breed a flue-cured variety of tobacco resistant to blue mold, about 1000 seed collections of Nicotiana tabacum varieties were obtained from Mexico, Central America, and parts of South America (20, 21). All of these were tested with disappointing results, in that not only was a very low level of resistance found, but when some resistance did appear it was usually associated with other undesirable characters that made it unusable. Resistance apparently is dependent upon many genes and repeated attempts in both Australia and the United States to find resistance within the cultivated commercial species have convinced investigators that there is no N. tabacum variety that possesses sufficient resistance to hope to obtain satisfaction from intraspecific hybridizations.

Breeding work in more recent years has centered around interspecific crosses. As far

Table 2. ORGANIC FUNGICIDES USED TO CONTROL BLUE MOLD OF TOBACCO IN THE UNITED STATES IN THE YEARS 1945-1951, AND IN CANADA IN 1947 AND 1948.

States or Province	Material	Formula or dosage	Percent or number growers using	Results	Remarks
1945					
Fla.	Fermate D[a]	20%	----	Good	
	Fermate D	10%, containing 1% zinc sulfate	----	Good	As good in station tests as the 20% D.
Va.	Fermate S[b]	----	----	----	Preferred over cuprous oxide-oil because application less difficult.
1946					
Conn.	Fermate	Fermate now used by all large growers and most small ones. In experiments at the Station Fermate dust gave as good control as the spray.			
Fla.	Fermate D	20% in talc	----	Good	Continues to give satisfaction.
	zineb D	10% in talc	----	Good	Even better than Fermate.
Ky.	Fermate D	15% Fermate, 85% Pyrophyllite	----	Excellent	In Experiment Station beds.
	Fermate S	----	----	Excellent	
S. Car.	Fermate D	15% Fermate, 85% Pyrophyllite	----	Excellent	Approximately 20 tons of dust used by growers this season.
Va.	Fermate D Fermate S	At least 90% of growers who used Fermate reported beneficial results. Over 50% of growers in flue-cured areas and about 25% in dark and burley areas sprayed with Fermate. All very much pleased with results and plan to use next season.			
1947					
Fla.	Fermate D	20% in talc	----	Good	Same as for 1946.
	zineb D	10% in talc	----	Good	
Ga.	Fermate D	----	----	----	Twice the usual amounts needed because of prolonged attack; shortage of fungicides in Georgia this year.
	Fermate S	----	----	----	
	bismuth subsalicylate	----	----	----	
Md.	Fermate D	----	----	Good	Fermate scarce.
	Fermate S	----	----	Good	
N. Car.	Fermate S	----	----	----	
	bismuth subsalicylate S with alcohol spreader	----	----	Good	
	Dithane-14 S	----	----	----	Caused severe damage to plants.
Ontario, Canada	Fermate S	----	----	----	Very scarce.
	bismuth subsalicylate S	----	----	----	500 lbs packaged as trial for flue growers.
Pa.	bismuth subsalicylate	----	----	----	Used only in limited amounts; Fermate very scarce.
	Dithane Z-78	----	----	----	
S. C.	Fermate D & S	----	----	----	In short supply.
1948					
Conn.	Fermate S	1-50	90	Excellent	
	Dithane Z-78	1-48	1	Good	
Fla.	Fermate D	20% in talc	75	Satisfactory when used as recommended; 3 times/wk;	
	Parzate D	10% in talc	Tests	infection too slight to judge results.	
Ga.	Fermate D	15%	----		
	Fermate S	4-100	82	Almost perfect commercial control with sprays and dusts.	
	bismuth subsalicylate S Dithane Z-78 S Dimole (Fermate-salicylic acid mixture)		3	----	
Ky.	Prepared to use Fermate S or D or Dithane Z-78 D	----	----	----	Very little used because of very mild attack.
Md.	Fermate D	15%	70	Excellent	
	Fermate S	2, 4-100	5	Excellent	
	Other organics	----	Trace	----	Mostly tests; results variable, mostly good.
Mass.	Fermate D	20%	5	Good	PDB used occasionally along with
	Fermate S	2-100	75	Good	Fermate to eradicate fungus from infected beds.
N. Car.	Fermate D	15%			
	Fermate S	4-100			
	Dithane Z-78	3-100	46	Excellent	Excellent where applied properly.
Ontario, Canada	Fermate D	Mfr.	25	Good	PDB 3 lbs/100 sq yds also gave
	Fermate S	2-40	65	Good	good results when used properly.
	Benzyl salicylate (aerosol bomb)	----	Less than 1	Uncertain	Minor injury.
Pa.	Fermate S	----	65 or more	----	15% sprayed with Bordeaux 8-4-100 or fixed copper spray (2 lbs Cu/100 gals).
S. Car.	Fermate D	15%	95	Good to excellent	In tests Dithane Z-78 and Karbam Black show promise of being as good as Fermate. Parzate, though effective, has caused some injury to plants.
Tenn.	Fermate D	15%	Some	----	
	Fermate S	2-4-100	Most	Fair	
Va.	Fermate D	15%	10	Good	
	Fermate S	3-100	50	Good	
	Parzate D & S	----	Trace	Good	
W. Va.	Fermate D	----	40	Good	
	Fermate S	2-100	40	Good	
Wis.	Fermate S	----	7 farms	----	Used in experimental spraying; disease not found in survey of tobacco area.

State or Province	Material	Formula or dosage	Percent or number growers using	Results	Remarks
1949					
Conn.	Fermate S	----	95	Almost perfect	
Fla.	Fermate D	----	75	----	
Ga.	Fermate D	----	----	----	Considerable damage when diluents other than talc or Pyrophyllite used.
Mass.	Iron carbamate spray or sprinkle	----	----	----	
N. Car.	Fermate and Dithane Z-78	----	----	----	
S. Car.	Fermate	----	----	----	
1950					
Conn.	Fermate D	----	----	Occasional growers using reported satisfaction.	
	Fermate S	Start with 1-50, increase to 2	95	Almost perfect where applied twice a week. Fermate now standard in this State. Dithane just as good but growers are in habit of using Fermate.	
Fla.	Fermate D	20%	50	Good	Applied 3 times/wk, from 15-35 lbs/acre.
	Dithane Z-78 D	10%	20	Good	Dithane Z-78 and Parzate have slightly better physical properties than Fermate.
	Parzate D	10%	10	Good (some injury)	
Ky.	Fermate S	----	Very small	----	Not much needed, mild year. Disease gradually reducing in injury from year to year as it did between 1937-1944.
N. Car.	Fermate S	4-100	22	Good	All fungicides gave satisfactory control when properly used. There is a strong trend toward use of dusts instead of sprays.
	Dithane Z-78 S	3-100	1	Good	
	Parzate S	3-100	few	Good	
	Fermate D	15%	32	Good	
	Dithane Z-78 D	10%	6	Good	Talc or Pyrophyllite recommended by Agricultural Extension Service as filler with dust.
	Parzate D	10%	1	Good	
Pa.	Copper and Fermate S	8-4-100 2 lbs	85	Good	
	Zerlate or Parzate	----	5	Fair	
S. Car.	Fermate D	15%	about 50	Good	
	Z-78 D	10%	few	Good	
	Parzate D	10%	few	Good	
	Fermate S	4-100	few	Good	
	Z-78 S	3-100	few	Good	
	Parzate S	3-100	few	Good	
	5379 & 5400 S	1-100	None	Good	New experimental fungicide from Carbide & Carbon Chemical Corp.
Tenn.	zineb D	5%	2	Good	Good control with both ferbam and zineb at Experiment Station. Zineb dust preferred as could be applied faster than spray and with less labor.
Greenville	ferbam D	10%	5	Good	
Knoxville	zineb D	5%	1	Good	
Greenville	ferbam S	2-50	10	Good	
	ferbam S	3-100	10	Excellent	Prepared Fermate dusts not generally available. Few use PDB with good results.
Knoxville	zineb S	3-100	1	----	
Va.	ferbam D	15%	10	Excellent	
	ferbam S	3-100	80	Excellent	
1951					
Fla.	ferbam D	15.6%	40	Good	Excellence of zineb results probably due to better coverage. Applied 3 time/wk using from 15 to 35 lbs/acre.
	zineb D	6.5%	40	Excellent	
Md.	Fermate D	15%	50	Good	Hot dry weather starting soon after initial infection stopped disease, preventing serious losses.
	Fermate S	2-100	50	Good	
N. Car.	Fermate D	15%	30	Good	Attack later than usual. In Border Belt most of crop was set before the disease became active.
	Dithane Z-78 or Parzate D	10%	5	Good	
	Fermate S	4-100	27	Good	
	Dithane Z-78 or Parzate S	3-100	3	Good	
Tenn.	Fermate D	10%	25	Excellent	
	Dithane D	5%	5	Good	
	ferbam D	10%	15	Good	
	zineb D	5%	15	Good	
	Fermate S	4-100	40	Excellent	
	Dithane S	3-100	5	Good	
	ferbam S	76%, using 5 tbs/gal	Est. 20	Good	
	zineb S	65%, using 2 1/2 tbs/gal	Very few	----	
Va.	ferbam D	10%	15	Excellent	Weather conditions mostly favorable for control of blue mold; plenty of plants.
	zineb D	6%	few	Excellent	
	ferbam S	3-4/100	75	Excellent	
	zineb S	2-100	few	Excellent	

[a] D = dust

back as 1936 Smith-White et al. (86) tested 100 species collections for resistance and found that greatest resistance was found in native Australian species, the one showing the most promise being N. debneyi Domin. During the war period the results of several species crosses for blue mold resistance were lost owing to absence of some of the staff, but in 1948 the blue mold breeding program was reopened under the supervision of Harold W. Lea and at present species hybrids with blue mold resistance are being selected from an extensive collection (58). Also, the Commonwealth Scientific and Industrial Research Organization at Canberra has conducted extensive experiments, under the direction of O. H. Frankel, for the past 2 years on the blue mold problem.

The following is an extract from the Annual Report for 1956-1957 of the Commonwealth Scientific and Industrial Research Organization: "TOBACCO INVESTIGATIONS (a) Genetics: In the breeding programme for resistance to blue mould work has been concerned with the location and transfer of resistance to commercial varieties of tobacco. Hybrids have been obtained with N. goodspeedii, N. excelsior, N. debneyi, and N. megalosiphon. Backcrosses from N. goodspeedii and N. debneyi to N. tabacum are showing considerable promise, one line in particular combining high field resistance with large leaves, although the leaf is unsuitable for curing.

"In addition an attempt is being made to induce resistance in a commercial variety by chemical mutagens or radiation, combined with testing by artificial inoculation. Thirty-five thousand first generation plants from X-irradiated seed or ultraviolet treated pollen all proved highly susceptible. Testing of second generation plants is now in progress" (79).

In the United States Clayton (19) reported in 1945 that resistance to blue mold is greatly dependent upon the age of the plants. He found that N. debneyi became resistant after 3 to 4 weeks. In 1949 Kincaid (57) reported that as a result of interspecific crossing of N. tabacum (commercial cigar-wrapper tobacco variety Rg) with N. debneyi (highly resistant to downy mildew, black root rot) eventually progenies were obtained which, though not resistant to downy mildew in the plant bed, did have some resistance to leaf spotting in the field. This seemed to be due to a quality which they had in common with N. debneyi, namely, that instead of collecting in drops on the leaves, moisture spreads over the leaf surface as a thin film of water which dries quickly. It was discovered that Chileno Correntino, an introduced variety from Argentina, also has this characteristic, allowing for the possibility that this variety may have originated from a N. tabacum x N. debneyi cross.

According to Clayton (20) there are indications that transfer of resistance from a species like N. debneyi, which is distantly related to N. tabacum, "once established in the tobacco genome, can be used far more rapidly and with fewer complications involving type, yield, and quality, than is the case with any resistance found within the cultivated species."

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